

PCDD and PCDF Exposure and Levels in Humans in Germany

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For nonoccupationally exposed persons, the daily intake via food consumption has been calculated to be 0.35 pg/kg body weight per day for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) and 2.3 pg/kg body weight per day for TCDD equivalents (TEqs). As compared to food, other sources and pathways are of minor importance. Food of animal origin contributes most, although human exposure begins with atmospheric emissions depositing these compounds on plant surfaces. In the meantime, a possible additional body burden from cardboard containers for cow's milk and coffee filters has been practically excluded. Of the 210 existing PCDDs and PCDFs, only 15 2,3,7,8-substituted isomers with a characteristic congener pattern can be found in samples of human origin. In adipose tissue and milk samples, mean levels for 2,3,7,8-TCDD of 7.2 and 3.6 pg/g fat, respectively, and of 56 (range 18–122) and 30 (range 10–72) pg TEqs/g fat, respectively, were determined. Human data revealed a dependency of polychlorinated dibenzo-*p*-dioxins/polychlorinated dibenzofurans (PCDD/PCDF) levels on age. In human milk, levels became reduced with the number of children born to mothers and duration of breast-feeding period. The average daily intake for a breast-fed child has been calculated to be 17 pg 2,3,7,8-TCDD/kg body weight per day and 142 pg TEqs/kg body weight per day, respectively. Levels in adipose tissue of infants, even if breast fed, were distinctly lower compared to human milk. In human milk, adipose tissue, and whole blood, PCDD/PCDF concentrations have been found to be equal on a fat-weight basis. Liver fat accumulated PCDD/PCDF with an alteration in the congener distribution pattern, whereas brain, even on a fat-weight basis, showed the lowest concentrations. Elevated or even high levels were found in occupationally exposed persons working in special chemical plants or involved in specific processes. There are limited data suggesting slightly elevated PCDD/PCDF levels are due to long-term consumption of a large share of food produced near point sources with a heavy emission or ingestion of soil or dust from such areas.

Introduction

The findings of polychlorinated dibenzo-*p*-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in five human milk samples from Germany (1) caused public concern and questions about the safety of breast feeding and the exposure of humans to these toxic substances. As a consequence, a large amount of analytical data from samples of human origin, especially milk, was produced in Germany with data on primary and secondary sources taken into consideration. To identify the routes contributing to the PCDD/PCDF body burden of man, air and food samples were analyzed and considered along with factors influencing the degree of contamination from these two important pathways.

Levels in Nonoccupationally Exposed Humans

Human Milk Levels

In Table 1, results for 112 human milk samples (mean age of mothers, 30 years) analyzed in the Bundesgesundheitsamt (BGA, Federal Health Office) have been summarized, stating mini-

Table 1. PCDD/PCDF levels (pg/g fat) in human milk.*

Congener	Minimum	Maximum	Mean	Median
2,3,7,8-TCDF	0.2	12	2.5	2.1
2,3,7,8-TCDD	0.9	9.7	3.6	3.3
1,2,3,7,8-PeCDF	<0.2	3.4	1.0	0.9
2,3,4,7,8-PeCDF	5.7	49	20	19
1,2,3,7,8-PeCDD	2.3	35	12	12
1,2,3,4,7,8-HxCDF	2.5	18	7.9	7.2
1,2,3,6,7,8-HxCDF	2.5	24	7.8	7.1
2,3,4,6,7,8-HxCDF	0.2	11	3.0	2.6
Total HxCDFs	5.9	53	19	17
1,2,3,4,7,8-HxCDD	2.5	33	10	9.8
1,2,3,6,7,8-HxCDD	11	126	47	47
1,2,3,7,8,9-HxCDD	2.0	20	9.0	8.2
Total HxCDDs	16	178	66	65
1,2,3,4,6,7,8-HpCDF	2.5	71	8.4	7.0
1,2,3,4,6,7,8-HpCDD	10	161	51	44
OCDF	<0.2	35	1.6	0.5
OCDD	78	1,300	344	281
TEqs	10	72	30	29

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDF, tetrachlorodibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEq, TCDD equivalents.

**n* = 112 samples.

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mum, maximum, mean, and median values. TCDD equivalents [TEqs (2)] ranged from 10 to 72 pg/g milk fat, with a mean of 30 pg/g milk fat. For each congener and TEqs, the maximum/minimum ratios were relatively low, and for at least

Table 2. PCDD/PCDF in human milk and adipose tissue (infants and adults): contribution (%) to concentration of total PCDD/PCDF and TEQs.

Congener	Milk		Adipose tissue			
	PCDD/PCDF	TEQs	Adults		Infants	
	PCDD/PCDF	TEQs	PCDD/PCDF	TEQs	PCDD/PCDF	TEQs
2,3,7,8-TCDF	0.5	0.8	0.3	0.5	0.6	1.3
2,3,7,8-TCDD	0.7	12.2	0.8	13.0	0.4	8.9
1,2,3,7,8-PeCDF	0.2	0.2	0.0	0.0	0.1	0.2
2,3,4,7,8-PeCDF	3.9	34.5	4.2	35.9	2.9	35.4
1,2,3,7,8-PeCDD	2.3	20.8	2.3	19.3	1.8	22.1
1,2,3,4,7,8-HxCDF	1.5	2.7	1.6	2.7	2.1	4.4
1,2,3,6,7,8-HxCDF	1.5	2.6	1.8	3.0	1.1	2.4
2,3,4,6,7,8-HxCDF	0.6	1.0	0.5	0.8	0.4	0.8
Total HxCDFs	3.5	6.3	3.9	6.5	3.5	7.5
1,2,3,4,7,8-HxCDD	1.9	3.4	2.0	3.4	1.2	2.6
1,2,3,6,7,8-HxCDD	8.9	15.9	9.5	16.0	7.0	14.8
1,2,3,7,8,9-HxCDD	1.7	3.0	1.2	2.1	1.5	3.2
Total HxCDDs	12.4	22.2	12.7	21.5	9.7	20.6
1,2,3,4,6,7,8-HpCDF	1.6	0.3	2.1	0.4	3.1	0.7
1,2,3,4,6,7,8-HpCDD	9.6	1.7	10.8	1.8	9.7	2.1
OCDF	0.3	0.0	0.0	0.0	0.2	0.0
OCDD	65.0	1.2	63.0	1.1	67.2	1.4
PCDF	9.9	42.0	10.5	43.3	11.0	45.0
PCDD	90.1	58.0	89.5	56.7	89.0	55.0

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TEQ, TCDD equivalents; TCDF, tetrachlorodibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa.

90–95% of the samples, they were near 10. This also can be clearly seen from Figure 1, which shows the distribution of TEQs and congeners such as 2,3,7,8-tetrachlorodibenzo-dioxin (TCDD), 2,3,4,7,8-pentachlorodibenzofuran (PeCDF), 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin (PeCDD), total hexa-

chlorodibenzodioxin (HxCDD), and octachlorodibenzo-*p*-dioxin (OCDD). As the medians were always lower than the mean values, all levels were much more likely to represent populations that were log-normally distributed than normally distributed (3).

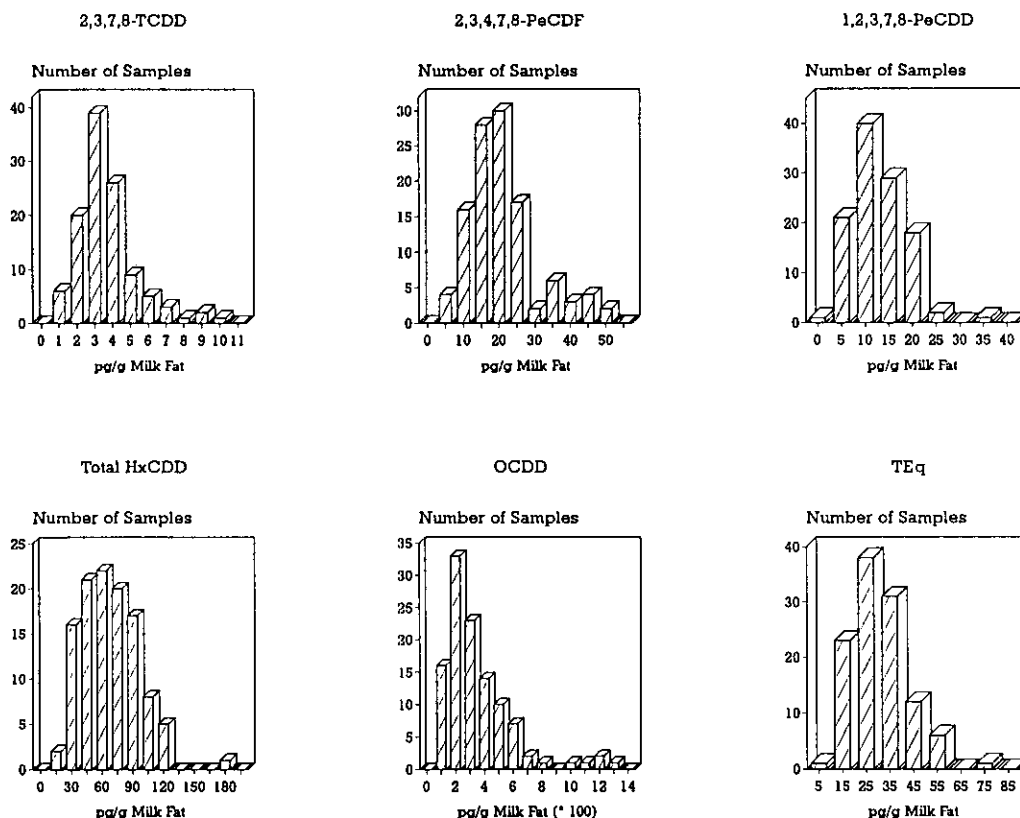


FIGURE 1. Distribution of polychlorinated dibenzo-*p*-dioxin/polychlorinated dibenzofuran levels in human milk.

Table 3. Mean PCDD/PCDF concentrations (pg/g fat) in human milk from different regions of Germany.

Congener	Berlin (n=53)	Weiden (n=14)	Rheinfelden (n=11)	Recklinghausen (n=10)	Flensburg (n=6)	Rastatt (n=12)
2,3,7,8-TCDF	2.2	3.3	5.9	1.4	1.6	1.8
2,3,7,8-TCDD	3.5	3.7	4.3	3.9	4.1	3.2
1,2,3,7,8-PeCDF	0.9	1.1	1.3	0.7	0.6	1.1
2,3,4,7,8-PeCDF	20	22	22	21	25	17
1,2,3,7,8-PeCDD	14	12	14	12	12	8.1
1,2,3,4,7,8-HxCDF	8.0	7.4	10	7.7	8.7	6.6
1,2,3,6,7,8-HxCDF	7.5	7.5	9.3	9.2	8.4	6.4
2,3,4,6,7,8-HxCDF	2.8	3.3	3.9	3.1	2.6	2.8
Total HxCDFs	18	18	23	20	20	16
1,2,3,4,7,8-HxCDD	11	9.3	12	13	7.3	7.1
1,2,3,6,7,8-HxCDD	52	44	51	46	46	28
1,2,3,7,8,9-HxCDD	9.4	9.6	11	8.3	8.3	6.7
Total HxCDDs	72	63	74	67	61	42
1,2,3,4,6,7,8-HpCDF	7.5	8.7	11	13	9.3	6.7
1,2,3,4,6,7,8-HpCDD	50	60	81	45	41	37
OCDF	1.8	0.8	1.5	4	0.3	0.7
OCDD	417	264	438	260	200	215
TEQs	31	30	34	30	31	22

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TEQ, TCDD equivalents; TCDF, tetrachlorodibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa.

Of the 210 existing PCDDs/PCDFs, only 15 are found in samples of human origin. This is remarkable, because in emissions and environmental samples a multitude of other congeners, some of them in relatively high concentrations, are also found. All congeners present in samples of human origin have a 2,3,7,8-chlorine substitution of the dibenzodioxin and -furan. Moreover, the tetra to hexachloro 2,3,7,8-substituted congeners are among the most toxic ones. 1,2,3,7,8,9-HxCDF could not be detected in these samples. 1,2,3,7,8-PeCDF and OCDF in most cases were found to be at levels near the limits of detection, and 1,2,3,4,7,8,9-HpCDF could not be identified.

The sum of all PCDDs was about 10 times higher than the sum of all PCDFs. Only among the pentachloro compounds was the level of the furans higher than that of dioxins. HpCDD, HpCDF, OCDF, and OCDD, representing the congeners of lower toxicity, make up 77% of all PCDDs/PCDFs. Contributions to the TEQs were made by 2,3,4,7,8-PeCDF with 35%, total HxCDD with 22%, 1,2,3,7,8-PeCDD with 21%, and 2,3,7,8-TCDD with 12%, although, as an example, the last congener had a share of <1% in the sum of all PCDDs/PCDFs. For further details see Table 2.

The levels of PCDDs increased with an increasing degree of chlorination. The main contributing PCDF congener was found to be 2,3,4,7,8-PeCDF. Furthermore, a strong conformity of the isomer distribution pattern for all the samples (especially for congeners with the same or nearly the same degree of chlorination) was evident (4,5) as demonstrated by the constant ratios for 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDD, 1,2,3,4,7,8-/1,2,3,6,7,8-HxCDFs and 1,2,3,4,6,7,8-HpCDD/OCDD.

Data on milk and adipose tissue samples from the same mothers are not available. Nevertheless, it can be presumed that on a fat-weight basis, PCDD/PCDF levels should be similar because practically identical concentrations in milk and adipose tissue samples (both on a fat-weight basis) from the same mothers were found (6) with other organochlorine compounds (hexachlorobenzene, β -hexachlorocyclohexane, DDT, DDE, PCB). Additional support for this assumption is provided by results for adipose tissue samples from eight women of child-

Table 4. Mean decrease (%) of PCDD/PCDF concentrations in human milk within one breast-feeding period.*

Congener	Weeks after delivery	
	6	12
2,3,7,8-TCDD	15	25
2,3,4,7,8-PeCDF	5	20
1,2,3,7,8-PeCDD	10	20
Total HxCDFs	10	10
Total HxCDDs	25	30
1,2,3,4,6,7,8-HpCDF	15	5
1,2,3,4,6,7,8-HpCDD	30	20
OCDD	45	40
TEQs	15	25

*Based on concentrations in milk samples of the first week.

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEQ, TCDD equivalents.

bearing age (7) with reported adipose levels of 2,3,7,8-TCDD, 2,3,4,7,8-PeCDF, and OCDD close to those found in milk (Table 1).

Dependency of PCDD/PCDF Levels on Various Parameters

Based on information gained from the mothers by means of a questionnaire, we examined several parameters likely to influence the PCDD/PCDF body burden.

Regional Differences. In Table 3 (8), the regional distribution of the samples (compare Table 1) is shown [Berlin, a large city with many types of emission; Weiden, a rural area; Rheinfelden, a town in a rural area but near a PCP-producing plant that is now closed; Recklinghausen, a town in an industrial area; Flensburg, a town at the Baltic Sea, where a high consumption of fish may be presumed; Rastatt, a town in a moderately industrialized area with a high contamination site (9) (for a discussion of this site see "Blood" under "Levels in Occupationally Exposed Persons")]. Due to the limited number of samples investigated, the results (mean values) are not representative. However, the

samples indicate that there has been no evident dependency of the PCDD/PCDF concentrations on the geographic residence of subjects. This statement remains true when considering levels established by other laboratories in samples from other regions of Germany (5,10–15). Moreover, these data underline the homogeneous pattern of distribution in general. In a multivariate statistical approach, Lindstrom et al. (16), who analyzed milk samples from North-Rhine Westphalia (Germany), found no correlation, irrespective of whether the mothers lived in urban areas or in a rural environment. Additionally, results obtained with samples from other countries with a similar degree of industrialization, compiled by the World Health Organization [WHO (17)], were in good agreement with those from Germany, as was also reported by Schecter et al. (18).

To produce a survey of PCDD/PCDF levels in Germany, supplemental results were added to those published by Fürst et al. (11), Ende (12), and Beck et al. (8) and combined with those for 728 samples. This representative overview (19) demonstrates the presence of a range from 6 to 87 pg TEQ/g milk fat, with a mean of 31 pg TEQ/g milk fat.

Number of Children. Figure 2 clearly shows a decrease of PCDD/PCDF levels with the number of children for 2,3,7,8-TCDD, 2,3,4,7,8-PeCDF, 1,2,3,7,8-PeCDD, all HxCDDs,

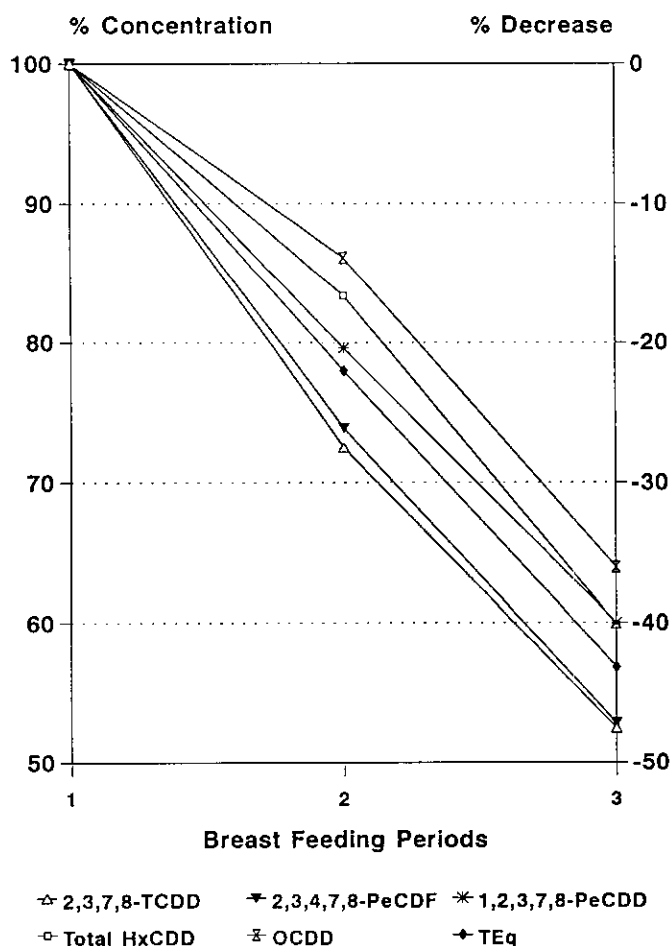


FIGURE 2. Relative levels of polychlorinated dibenzo-*p*-dioxins/polychlorinated dibenzofurans in human milk versus number of breast feeding periods (first child, 100%).

Table 5. PCDD/PCDF concentrations (pg/g fat) in samples of adipose tissue from nonoccupationally exposed humans.*

Congener	Minimum	Maximum	Mean
2,3,7,8-TCDF	0.7	6.0	2.5
2,3,7,8-TCDD	1.5	18	7.2
1,2,3,7,8-PeCDF	0.1	1.6	0.4
2,3,4,7,8-PeCDF	10	101	40
1,2,3,7,8-PeCDD	8.8	48	21
1,2,3,4,7,8-HxCDF	4.8	39	15
1,2,3,6,7,8-HxCDF	4.7	47	16
2,3,4,6,7,8-HxCDF	2.1	10	4.7
Total HxCDFs	12	96	36
1,2,3,4,7,8-HxCDD	8.7	29	19
1,2,3,6,7,8-HxCDD	35	129	89
1,2,3,7,8,9-HxCDD	3.8	20	12
Total HxCDDs	48	178	119
1,2,3,4,6,7,8-HpCDF	7.2	35	20
1,2,3,4,6,7,8-HpCDD	39	216	101
OCDF	0.1	0.8	0.4
OCDD	212	1,061	591
TEQs	18	122	56

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEQ, TCDD equivalents.

*n=20 samples.

Table 6. Definition of PCDD/PCDF background concentrations (pg/g) in human fat.

Compound	Range
TCDF	< 1 – 10
TCDD	2 – 20
Total PeCDFs	2 – 100
PeCDD	2 – 50
Total HxCDFs	2 – 100
Total HxCDDs	10 – 300
Total HpCDFs	2 – 50
HpCDD	10 – 250
OCDF	< 1 – 20
OCDD	50 – 1500
TEQs	6 – 140

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEQ, TCDD equivalents.

OCDD, and TEQs. For these congeners and TEQs, the concentrations in milk from mothers with their third child decreased to between 36 and 47% (43% for TEQs) as compared to milk from mothers with their first child. Lindstrom et al. (16) and Fürst et al. (5,15) found similar results for another cohort.

Duration of Breast-Feeding Period. Three samples of milk from 15 mothers with their first child were collected during the breast-feeding period (sample 1, 1st week; sample 2, 6th week; sample 3, 12th week) and were analyzed in parallel clean-ups. As a result, different rates of decrease in the levels of the congeners were ascertained. Higher chlorinated PCDDs/PCDFs were eliminated faster than lower chlorinated congeners. Also, PCDDs were eliminated more rapidly than PCDFs. The main decrease was found during the first weeks. In part, the individual data revealed distinct deviations from the means summarized in Table 4. Based on the concentrations in milk from the first week, we found a decline for TEQs of 25% and for OCDD of 40%

Table 7. Daily intake of PCDDs/PCDFs (pg/kg body weight per day) by infants via human milk.^a

	Minimum	Maximum	Mean
2,3,7,8-TCDD	4	48	17
TEqs	48	346	142

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; TEq, TCDD equivalents.

^aBody weight 5 kg; milk quantity: 800 ml; fat content: 3%. See Table 1 for levels in milk.

(20). Similar results were reported by Fürst et al. (15): One mother breast-feeding for 1 year retained 28% of TEqs from her first-week levels.

Age. Figure 3 suggests a dependency of the 2,3,7,8-TCDD and TEq levels on the age of the mother with her first child. There was an increase of 0.12 pg TCDD/g milk fat per year and 0.71 pg TEq/g milk fat per year. For this parameter we had selected only mothers with their first child to avoid lower levels in milk from mothers with more births. In principle, human fat and blood samples are more suitable for such a study because of the relatively short time that women are of childbearing age and different factors influencing the levels in human milk. However, in

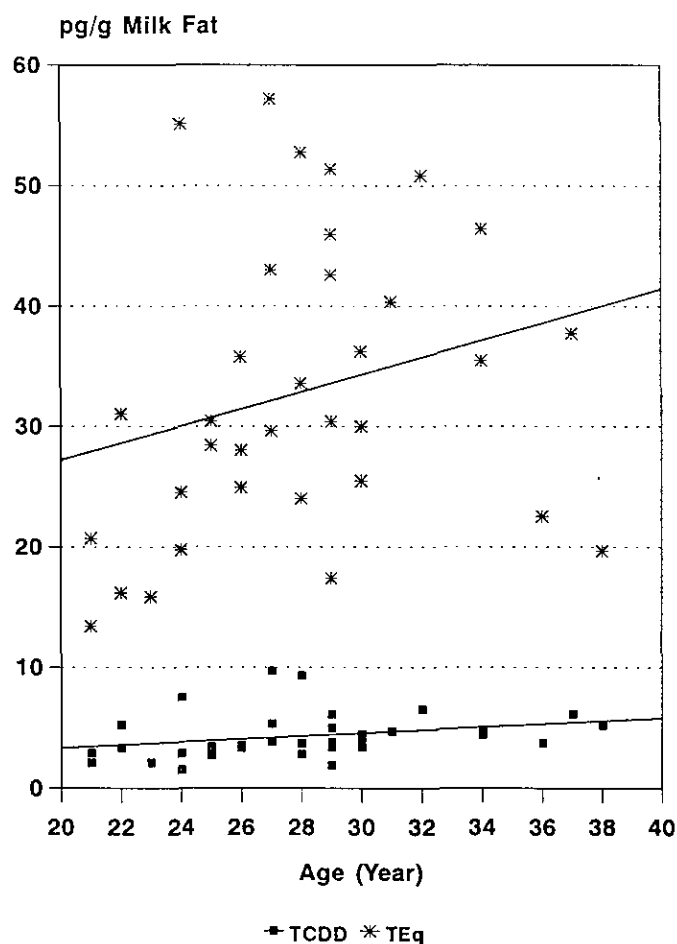


FIGURE 3. Tetrachlorodibenzo-*p*-dioxin (TCDD) and TCDD equivalent levels in human milk (first child) versus age.

Table 8. PCDD/PCDF levels (ppg/g fat) in adipose tissue of infants.

Congener	Infant					
	A	B	C	K	L	M
2,3,7,8-TCDF	<0.5	<0.5	<0.5	1.4	3.1	1.1
2,3,7,8-TCDD	1.0	0.7	<0.5	<0.2	<0.5	2.1
1,2,3,7,8-PeCDF	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
2,3,4,7,8-PeCDF	5.5	4.5	3.9	1.6	6.0	13
1,2,3,7,8-PeCDD	2.5	2.0	1.9	0.5	2.9	12
1,2,3,4,7,8-HxCDF	1.0	1.8	1.1	2.3	7.7	7.5
1,2,3,6,7,8-HxCDF	0.7	1.6	1.3	0.7	2.4	4.8
2,3,4,6,7,8-HxCDF	0.6	<0.5	<0.5	0.5	0.3	2.0
Total HxCDFs	2.3	3.7	2.7	3.5	10	14
1,2,3,4,7,8-HxCDD	1.7	1.3	0.8	0.3	2.6	6.2
1,2,3,6,7,8-HxCDD	9.1	6.0	5.7	2.1	13	37
1,2,3,7,8,9-HxCDD	1.5	1.6	0.9	0.8	3.6	7.1
Total HxCDDs	12	8.9	7.4	3.2	19	50
1,2,3,4,6,7,8-HpCDF	2.5	2.6	1.8	2.2	11	12
1,2,3,4,6,7,8-HpCDD	5.9	5.8	6.5	5.1	20	57
OCDF	<0.5	1.2	<0.5	<0.5	<0.5	<0.5
OCDD	55	43	81	59	120	341
TEqs	6.6	5.4	4.4	2.1	8.4	22
Age, months	9.3	3.8	4.8	4.8	6.2	23
Breast feeding, days						
Exclusively	21	6	0	0	90	56
Partially	63	0	0	0	0	28

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEq, TCDD equivalents.

the data sets of Lindstrom et al. (16) and Fürst et al. (5), a dependency on age was not found.

Smoking habits. Differences of PCDD/PCDF levels in human milk in relation to cigarette smoking could not be seen because of a lack of differences in concentrations not only for TEqs but also for individual congeners between both groups. As the concentrations in cigarettes have been reported to be 3.4 pg OCDD/cigarette and 0.1 pg TEq/cigarette (21), elevated TEq levels could not be expected theoretically even for long-time, heavy smokers if the intake from smoking was compared with the intake via food consumption.

Vegetarian Diet. The PCDD/PCDF levels in 6 milk samples from mothers with a vegetarian diet (TEq, 14.0 pg/g milk fat) were slightly lower than the levels in 60 milk samples from mothers with a common diet (TEq, 16.2 pg/g milk fat). The differences may have been so small because the vegetarian diet was taken only for a relatively short period and milk and milk products were not always excluded and are important sources for the PCDD/PCDF body burden.

Two samples from vegans (individuals who eat no meat or meat byproducts, including poultry and fish, no milk or other dairy products, and no eggs) were taken. These samples had 7.8 and 14.0 pg TEq/g milk fat and were included in the samples from the vegetarians.

Indoor Wood Paneling. Some years ago much of the wood used for construction was treated with pentachlorophenol (PCP) containing high levels of PCDDs/PCDFs, especially higher chlorinated ones with a predominance of OCDD. In some cases, this resulted in elevated indoor air PCDD/PCDF levels in houses furnished with wood paneling. Levels of milk samples from 37 mothers living in houses with wood paneling did not differ from samples from mothers without such paneling in their homes.

Table 9. Mean ratios of PCDD/PCDF concentrations in human liver fat and adipose tissue fat from adults (23) and infants (28).

Congener	Adults (n=28)	Infants (n=3)
2,3,7,8-TCDF	2.20	—
2,3,7,8-TCDD	2.05	1.1
2,3,4,7,8-PeCDF	4.93	2.6
1,2,3,7,8-PeCDF	1.22	1.2
Total HxCDFs	9.38	12.8
Total HxCDDs	1.76	1.3
1,2,3,4,6,7,8-HpCDF	15.42	9.5
1,2,3,4,6,7,8-HpCDD	9.39	8.5
OCDF	7.43	5.3
OCDD	11.83	14.1

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa.

Even for OCDD, levels did not differ between those exposed and those not exposed.

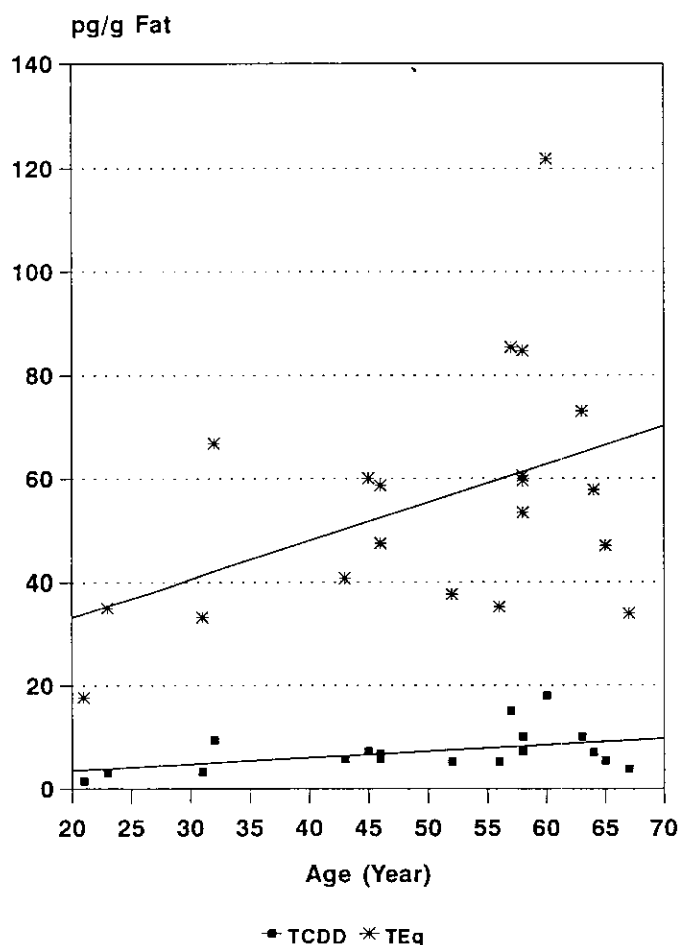
For one milk sample (TEq, 36; OCDD, 257 pg/g milk fat) with levels near the mean values for the reference group (Table 1), the corresponding indoor air PCDD/PCDF levels were measured to be nearly 2 pg TEq/m³ and 200 pg OCDD/m³, respectively, con-

Table 10. PCDD/PCDF concentrations (pg/g fat in adipose tissue of occupationally exposed workers.*

Congener	Minimum	Maximum	Mean
2,3,7,8-TCDF	0.5	4.9	1.5
2,3,7,8-TCDD	5.9	2,252	225
1,2,3,7,8-PeCDF	0.2	5.8	1.3
2,3,4,7,8-PeCDF	14	205	67
1,2,3,7,8-PeCDD	12	605	126
1,2,3,4,7,8-HxCDF	4.8	1,038	191
1,2,3,6,7,8-HxCDF	5.0	365	95
2,3,4,6,7,8-HxCDF	1.2	43	10
Total HxCDFs	11	1,431	296
1,2,3,4,7,8-HxCDD	9.3	1,133	204
1,2,3,6,7,8-HxCDD	40	7,631	1,017
1,2,3,7,8,9-HxCDD	5.8	849	163
Total HxCDDs	55	9,613	1,384
1,2,3,4,6,7,8-HpCDF	6.1	935	181
1,2,3,4,7,8,9-HpCDF	0.2	25	5.4
Total HpCDFs	6.3	947	186
1,2,3,4,6,7,8-HpCDD	24	4,120	747
OCDF	0.5	33	1.9
OCDD	302	15,892	3,264
TEqs	37	2,928	502

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEq, TCDD equivalents.

*n=57 samples.

**FIGURE 4.** Tetrachlorodibenzo-*p*-dioxin (TCDD) and TCDD equivalent levels in human adipose tissue versus age.

centrations showing elevated indoor air levels (W. Rotard, unpublished results).

Other Parameters. A negative correlation was found between PCDD/PCDF levels in human milk and the child weight (16) and for the difference of weight from the optimum weight of the mothers (5). No such correlation was recognized for the fat content of milk and mother's body fat status (16).

Adipose Tissue

Adults. In Table 5, the results of the analysis of 20 samples of human adipose tissue (males, mean age, 50 years) have been summarized (22). For TEqs, a range of 18–122 pg/g, with a mean of 56 pg/g fat was found. Compared with levels in human milk, these levels were distinctly higher. This was partly due to a higher age of donors of adipose tissue, but also due to different parameters diminishing the PCDD/PCDF levels in human milk such as the duration of breastfeeding period and the number of children. The isomer patterns characterized by the percentage of congeners as referred to total PCDD/PCDF were without any evident differences (Table 2).

Figure 4 shows a similar dependency of PCDD/PCDF levels on age as described for milk samples because there was a nearly identical increase of 0.12 pg TCDD/g fat per year and 0.77 pg TEq/g fat per year, respectively.

Thoma et al. (23–25) reported on 76 samples of adipose tissue from humans of different age, sex, body weight, height, and health status with similar PCDD/PCDF levels. No correlation of levels with age or sex could be seen. Considering all data in combination with that of countries with a similar degree of industrialization, we defined the ranges listed in Table 6 as "PCDD/PCDF background levels" in humans from industrialized countries at the present time (22).

Infants. As the PCDD/PCDF levels in human milk have been stated to be higher than in food (26,27) and infant formula (27),

differences in levels might be expected for adipose tissue samples from breast-fed and nonbreast-fed infants. To examine the suspected differences, adipose tissue samples from six infants who had died from sudden infant death syndrome (SIDS) were analyzed (28). The choice of SIDS samples was based on the fact that the nutritional status of the children affected is entirely normal, which is not always true of sick children.

The average daily intake of a breast-fed child has been calculated to be 17 pg 2,3,7,8-TCDD/kg body weight per day and 142 pg TEQ/kg body weight per day (29), (for further details, see Table 7) and exceeds the upper limit of the tolerable daily intake [1–10 pg TEQ/kg body weight per day, fixed by the Bundesgesundheitsamt (BGA) (30)] by a factor of more than 10. Although for risk assessment the daily intake over the entire lifetime has to be considered (30,31), the short-term intake of levels exceeding the toxicological limits during breast-feeding is of concern both because of the long half-life of some of these compounds and also in light of prospective health care. However, according to the BGA, no adverse health effects in breast-fed children have been demonstrated and because the advantages of breastfeeding for the first 4–6 months of life outweigh the theoretical risks, breastfeeding is encouraged in accordance with WHO (31) recommendations.

By feeding with infant formula, the daily intake is estimated to be lowered by a factor of 1/20. Therefore, the PCDD/PCDF levels in adipose tissue samples of breast-fed infants are surprisingly low, although they are higher than in nonbreast-fed infants (Table 8). In any case, for infants it has to be considered that adipose tissue weight increases more strongly than body weight with age, but this small database does not permit a calculation of the extent of prenatal exposure and the extent of absorption from the gastrointestinal tract (7,32,33). The isomer pattern characterized by the percentage of congeners as compared with total PCDD/PCDF was without any important differences to human milk or adipose tissue samples (see Table 2). This is in contrast to findings by Thoma (23,25), who reported the presence of relatively higher levels, especially of TCDD, in eight

infants. As there are no data on the causes of death, elevated levels (e.g., because of cachexia) cannot be excluded. Nevertheless, these mean levels for all congeners except TCDD were lower than the corresponding ones for human milk.

In contrast, Bowman's (34) investigations in rhesus monkeys have shown 2,3,7,8-TCDD levels in the fat of the offsprings (breast-fed for 4 months) were more than four times higher than in the fat of the mothers with a 5 and 25 pg/g TCDD level. Assuming TCDD distribution across the placenta at equal tissue concentration, the lactational transfer led to a 3.3 bioconcentration of TCDD in infant fat compared to that in mother's fat.

Blood

As sampling of adipose tissue involves a surgical intervention and human milk is restricted to breast-feeding mothers, blood sampling is less invasive and more generally available. A disadvantage is that investigations involving blood are much more complicated because of the low levels found. As a basic requirement, it is important to know the relation of PCDD/PCDF concentrations in blood and adipose tissue, especially to recognize specific exposure situations.

Measurements of adipose tissue and whole blood of three persons revealed a good conformity of the results on a lipid weight basis for each person (35). Additionally, the PCDD/PCDF levels of 10 and 85, respectively, whole blood samples of nonexposed persons (36,37) corresponded to those for adipose tissue (Table 5), both on a lipid-weight basis. In another study, it was shown that higher chlorinated compounds such as OCDD in plasma lipid were present in higher amounts than in adipose fat, but whole blood lipid and adipose fat values for the higher chlorinated as well as lower chlorinated values were about equal (38,39).

Organs

Liver. Adipose tissue and liver samples from 28 adults of different age, sex, body weight, height, and health status were analyzed by Thoma et al (23). In addition, the results of the analysis of three liver samples from three SIDS infants with corresponding adipose tissue samples were published (28). Both studies have shown that liver/adipose tissue ratios (both on a fat-weight basis) for HxCDFs, HpCDF, HpCDD, and OCDD are strikingly high, whereas for the other congeners the ratios were lower (Table 9). This suggests distinct differences in the PCDD/PCDF pattern in liver in comparison to adipose tissue (40,41). Contrary to the results based on fat weight, liver was found not to accumulate TEQs if results were expressed on a whole weight basis (28).

Thymus, Spleen, and Brain. From the three infants who had died from SIDS, thymus, spleen, and brain (gray and white matter) were also analyzed. In comparison to adipose tissue, the levels were low in brain, even on a fat-weight basis. This means that there is no accumulation in the brain. For TEQs in thymus and spleen on a fat-weight basis, the ratios to adipose fat are in many cases above 2, but on a whole weight basis, none of these organs was found to accumulate PCDDs/PCDFs (28).

Placenta. Concentration ratios for placental and adipose tissue (both on a fat-weight basis) of eight women increased in the group of PCDDs/PCDFs with increasing degrees of chlorination (concentration ratios placenta/adipose tissue: TCDD, 0.4;

Table 11. PCDD/PCDF concentrations (pg/g fat) in adipose tissue and spinal cord of one person.

Congener	Adipose tissue	Spinal cord
2,3,7,8-TCDF	0.6	<4.5
2,3,7,8-TCDD	56	13
1,2,3,7,8-PeCDF	1.3	1.8
2,3,4,7,8-PeCDF	32	3.1
1,2,3,7,8-PeCDD	47	6.1
1,2,3,4,7,8-HxCDF	73	7.8
1,2,3,6,7,8-HxCDF	44	3.5
2,3,4,6,7,8-HxCDF	3.7	1.0
Total HxCDFs	121	12
1,2,3,4,7,8-HxCDD	67	7.1
1,2,3,6,7,8-HxCDD	349	22
1,2,3,7,8,9-HxCDD	89	7.1
Total HxCDDs	505	36
1,2,3,4,6,7,8-HpCDF	58	7.9
1,2,3,4,6,7,8-HpCDD	186	12
OCDF	0.8	2.5
OCDD	1,004	144
TEQs	161	23

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEQ, TCDD equivalents.

Table 12. PCDD/PCDF concentrations (pg/g fat) in food samples of animal origin.

Congener	Cow's Milk	Butter	Pork	Beef	Sheep	Chicken	Eggs	Herring	Cod	Redfish
2,3,7,8-TCDF	0.7	0.15	0.11	0.3	0.6	2.1	1.1	57	98	78
2,3,7,8-TCDD	0.2	0.08	0.03	0.6	0.01	0.3	0.2	4.7	23	2.8
1,2,3,7,8-PeCDF	0.2	0.09	0.01	0.01	0.01	0.01	0.6	16	48	31
2,3,4,7,8-PeCDF	1.4	0.45	0.08	1.5	0.9	1.5	0.8	29	3.1	25
1,2,3,7,8-PeCDD	0.7	0.41	0.12	0.8	0.5	0.7	0.4	12	1.3	6.5
1,2,3,4,7,8-HxCDF	0.9	0.43	0.15	0.8	0.9	0.6	0.4	3.0	6.9	3.5
1,2,3,6,7,8-HxCDF	0.8	0.44	0.07	0.6	1.2	0.4	0.3	4.2	13	6.0
2,3,4,6,7,8-HxCDF	0.7	0.31	0.05	1.3	1.5	0.3	1.7	3.6	8.2	7.2
Total HxCDFs	2.4	1.18	0.27	2.7	3.6	1.3	2.4	11	28	17
1,2,3,4,7,8-HxCDD	0.3	0.15	0.21	0.6	0.3	0.5	1.3	1.2	0.01	0.5
1,2,3,6,7,8-HxCDD	1.1	0.95	0.29	1.9	1.5	2.8	1.4	5.8	17	8.4
1,2,3,7,8,9-HxCDD	0.4	0.26	0.06	0.6	0.4	0.6	0.5	1.0	5.2	1.3
Total HxCDDs	1.8	1.36	0.56	3.1	2.2	3.9	3.2	8.0	22	10
1,2,3,4,6,7,8-HpCDF	0.5	0.34	1.1	2.2	8.1	0.8	0.6	1.6	10	1.5
1,2,3,4,6,7,8-HpCDD	2	1.5	2.1	18	15	6.0	0.4	3.6	10	3.0
OCDF	1	0.25	0.41	0.2	0.3	0.6	0.2	1.4	2.1	0.3
OCDD	10	3.4	19	25	68	52	12	19	83	11
TEqs	1.79	0.81	0.28	2.59	1.65	2.25	1.52	33.7	42.7	30.7

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzo-*p*-dioxin; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEq, TCDD equivalents.

Table 13. Average daily intake (pg/d) for 2,3,7,8-TCDD and TEqs via food consumption (26).

Food	Consumption, g/day	2,3,7,8-TCDD	TEq
Meat/meat products	38 (fat)	7.0	35.9
Milk/milk products	33 (fat)	6.2	55.1
Eggs	3.9 (fat)	0.8	5.9
Fish/fish products	1.8 (fat)	8.6	60.5
Vegetable oil	26 (fat)	0.1	0.4
Vegetable	244 (wet weight)	1.2	3.7
Fruit	130 (wet weight)	0.7	2.0
Total	102.7 (fat)	24.6	164

Abbreviations: TCDD, tetrachlorodibenzo-*p*-dioxin; TEq, TCDD equivalents.

OCDD, 1.6). Because fat content of the placental tissue ranged from 0.6 to 1%, on a whole weight basis this tissue is low in these substances compared to adipose tissue (7).

Levels in Occupationally-Exposed Persons

Adipose Tissue

In a chemical plant in Ludwigshafen, Germany, an incident occurred in 1953 in the trichlorophenol manufacturing plant leading to a high contamination by 2,3,7,8-TCDD. More than 30 years later, Schecter et al. (42) analyzed six samples of adipose tissue from workers who had been exposed to this compound. For 2,3,7,8-TCDD the range was between 14 and 141 (mean, 49) pg/g fat, i.e., reaching from normal background levels (see Table 6) to elevated levels. However, even the minimum level may have been elevated at the time of exposure. The other congeners were in the normal range.

Rappe et al. (43) analyzed four samples of adipose tissue from persons who had worked at the same plant. High levels (>200 pg/g), and a mean value of 150 pg/g for 2,3,7,8-TCDD distinctly exceeding the background contamination could be identified. Other congeners were in the normal range.

Table 14. PCDD/PCDF levels (pg/g fat) in geese fat tissue.*

Congener	Sample					
	1	2	3	4	5	6
2,3,7,8-TCDF	3.2	2.4	3.1	<0.2	<0.2	<0.2
2,3,7,8-TCDD	5.8	5.8	6.0	0.2	0.2	0.2
1,2,3,7,8-PeCDF	4.7	4.1	7.9	0.3	0.3	0.2
2,3,4,7,8-PeCDF	8.6	5.9	8.0	0.4	0.4	0.3
1,2,3,7,8-PeCDD	10	11	11	0.4	0.5	0.5
1,2,3,4,7,8-HxCDF	7.9	5.6	13	0.5	0.5	0.2
1,2,3,6,7,8-HxCDF	6.3	3.7	8.1	0.3	0.3	0.2
2,3,4,6,7,8-HxCDF	1.8	2.3	3.5	0.2	0.2	0.2
Total HxCDFs	16	12	24	1.0	1.0	0.6
1,2,3,4,7,8-HxCDD	2.9	4.5	4.1	0.4	0.3	0.2
1,2,3,6,7,8-HxCDD	7.2	9.0	8.2	1.5	1.6	0.3
1,2,3,7,8,9-HxCDD	1.5	2.3	2.3	0.4	0.3	0.2
Total HxCDDs	12	16	15	2.3	2.3	0.7
1,2,3,4,6,7,8-HpCDF	4.1	3.5	6.3	1.1	0.8	0.2
1,2,3,4,6,7,8-HpCDD	3.4	6.4	5.4	2.6	2.3	0.5
OCDF	0.6	1.0	1.5	0.5	0.6	0.2
OCDD	9.4	23	18	<10	<10	<10
TEqs	19	18	20	1.0	1.0	0.8

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzo-*p*-dioxin; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEq, TCDD equivalents.

*Samples 1-3, contaminated area; samples 4-6, background.

Another company in Germany with two chemical plants in Ingelheim and Hamburg produced, in addition to other substances, lindane, chlorophenols, and 2,4,5-T in series of combined reactions with thermal decomposition, resulting in the formation of a multitude of PCDD/PCDF isomers including non-2,3,7,8-substituted ones. From 57 employees of these two plants, adipose tissue samples were taken. The ranges of PCDD/PCDF concentrations in these samples are given in Table 10, showing that the lower concentrations were similar to those found in human milk or adipose tissue from nonexposed persons (22). Fourteen samples had TEq levels within the background range (see Table 6). The maximum levels for PCDD/PCDF congeners were extremely high in some persons. In 10 samples TEq levels exceeded

Table 15. PCDD/PCDF levels (pg/g wet weight) in kale.*

Congener	Sample				
	1	2	3	4	5
2,3,7,8-TCDF	0.07	0.16	0.84	1.21	3.05
Other TCDFs	1.43	5.30	14.12	37.80	62.45
2,3,7,8-TCDD	<0.01	0.05	0.05	0.10	0.14
Other TCDDs	1.07	3.93	6.89	10.86	17.79
1,2,3,7,8-PeCDF	0.14	0.39	1.03	1.00	1.15
2,3,4,7,8-PeCDF	0.07	0.14	0.46	0.28	0.44
Other PeCDFs	2.06	5.67	13.37	13.32	15.22
1,2,3,7,8-PeCDD	0.26	0.06	0.23	0.15	0.16
Other PeCDDs	1.44	1.85	5.58	6.15	5.42
1,2,3,4,7,8-HxCDF	0.07	0.09	0.26	0.07	0.12
1,2,3,6,7,8-HxCDF	0.07	0.09	0.27	0.06	0.09
2,3,4,6,7,8-HxCDF	<0.01	<0.01	0.02	0.06	0.09
Other HxCDFs	1.36	0.91	2.62	0.67	0.82
1,2,3,4,7,8-HxCDD	0.09	0.02	0.11	0.04	0.08
1,2,3,6,7,8-HxCDD	0.07	0.10	0.14	0.08	0.11
1,2,3,7,8,9-HxCDD	0.08	0.07	0.14	0.05	0.09
Other HxCDDs	3.84	1.06	2.38	3.52	2.42
1,2,3,4,6,7,8-HpCDF	0.17	0.35	1.08	0.14	0.20
1,2,3,4,6,7,8-HpCDD	0.33	0.62	2.26	0.32	0.88
1,2,3,4,6,7,9-HpCDD	0.36	0.58	2.75	0.42	1.14
OCDF	0.11	0.24	0.52	0.08	0.18
OCDD	1.29	3.01	0.68	1.20	2.33
TEqs	0.22	0.24	0.66	0.53	0.87

Abbreviations: PCDD, polychlorinated dibenzo-*p*-dioxin; PCDF, polychlorinated dibenzofuran; TCDD, tetrachlorodibenzo-*p*-dioxin; Pe, penta; CDF, chlorodibenzofuran; CDD, chlorodibenzo-*p*-dioxin; Hx, hexa; Hp, hepta; O, octa; TEq, TCDD equivalents.

*Samples 1–2, background; samples 3–5, contaminated areas.

1000 pg/g fat, four of those samples showed 2,3,7,8-TCDD concentrations above 1000 pg/g fat. In all samples only 2,3,7,8-substituted congeners could be found. 2,3,7,8-TCDF and OCDF were only found in concentrations within the range of background contamination. PeCDF levels were relatively low, whereas those of 2,3,7,8-TCDD and HxCDDs were especially high. In some samples only 2,3,7,8-TCDD was found at an elevated concentration. These latter samples had been taken from workers involved in the 2,4,5-T production only. Employees who had contact with the chemical wastes (thermal destruction residues) from the other production processes had, however, been exposed to a multitude of PCDD/PCDF isomers (and other organochlorine substances), leading to elevated levels for many of the congeners (see Table 10).

With regard to chloracne, it is important to state that chloracne is not a reliable indicator of PCDD/PCDF exposure because in a few cases chloracne was associated with relatively low PCDD/PCDF concentrations in the body fat, whereas other workers with higher concentrations (>1000 pg TEq/g) did not develop chloracne (22).

As times of exposure and times of sampling sometimes differed, estimated PCDD/PCDF levels at the time of exposure, using half-life value extrapolation, suggested extremely high concentrations (up to 12,000 pg 2,3,7,8-TCDD/g adipose fat) for some samples (22,24).

Blood

PCDD/PCDF concentrations in whole blood of 85 persons involved in three fire incidents with considerable PCDD/PCDF

emissions were reported by Pöpke et al. (44). These persons had been engaged in the clean-up activities after the fire or had been employed at the fire site. Their PCDD/PCDF levels showed no evident relation to the exposure because levels and congener pattern were similar to the samples with background contamination.

The same authors (35,45) analyzed three blood samples. Two of them were from females who had worked for 13 years in a kindergarten in which the indoor wood paneling had been treated with preservatives containing PCP. The third sample was collected from a carpenter who had sprayed PCP-containing wood preservatives for 3–4 years in new houses. None of the samples had elevated levels compared to the background levels listed in Table 6. In conclusion, it can be stated that for these three samples a low-level, long-term indoor exposure or exposure from spraying PCP did not lead to a significant elevation of PCDD/PCDF concentrations. This is also true of the higher chlorinated PCDDs, which are the dominant by-products in the composition of PCP (35,45).

In blood samples from 32 persons living near, or working in, a metal reclamation plant in Rastatt, elevated levels were found in many of the plant workers (9). The concentrations of PeCDFs, HxCDFs, and HpCDFs were above the background levels, changing the usual human blood congener distribution pattern. Also, some samples from nonoccupationally exposed persons living in the contaminated area had comparable elevated levels for these congeners. This is possible due to vegetable consumption from kitchen gardens and possibly soil and/or dust ingestion being a pathway for exposure, especially for children. It seems that elevated PCDD/PCDF levels for nonoccupationally exposed persons in very rare cases are caused by long-term dust and soil ingestion or consumption of food produced near point sources with heavy PCDD/PCDF emission (9).

Spinal Cord

In Table 11, PCDD/PCDF levels in the spinal cord and adipose tissue (both on fat-weight basis) of an individual are compared (29). The levels in the spinal cord are distinctly lower even on a fat-weight basis, which documents that only a limited passage through the blood-brain barrier had taken place.

Exposure

The sources of PCDDs/PCDFs in the environment are numerous and will not be discussed here. Evaluations of different sources for Germany have been published by Hagenmaier (46,47) and Hutzinger (48,49).

The similarity of human PCDD/PCDF data for nonoccupationally exposed persons reveals that there is no evident dependency of levels on the geographical residence of subjects. These findings support the assumption that food intake is an important pathway for man because most food is not produced in the residential area of the consumers, rather food is of widespread origin. This is equivalent to generally uniform contamination of food with PCDDs/PCDFs. Contrary to this, it is known that PCDD/PCDF levels in air (50–55) differ between industrialized and rural zones up to a factor of 15 (55) and that even elevated levels in indoor air (45) do not significantly affect the body burden.

Food

On the basis of food samples summarized in Table 12 (26), the daily intake via food consumption was calculated to be 25 pg/person/day for 2,3,7,8-TCDD and 164 pg/person/day for TEQs (Table 13). A conversion to body weight (body weight 70 kg) has resulted in levels of 0.35 pg/kg body weight per day for 2,3,7,8-TCDD and 2.3 pg/kg body weight per day for TEQs. Most of the body burden caused by food consumption comes in equal shares from meat, milk, and fish. Food of vegetable origin is of minor importance, although the human exposure route begins with atmospheric emissions, including gaseous diffusion depositing these compounds on plant surfaces (56).

These results were confirmed on a broader basis of analytical results from samples of milk, cheese, butter, beef, veal, pork, lamb, chicken meat, canned meat, lard, fish, cod liver oil, salad oil, and margarine (27). Other data for milk, pork, beef, and fish samples were in the same range (57).

Fish samples were characterized by elevated concentrations of 2,3,7,8-TCDF and 2,3,4,7,8-PeCDF. Fourteen samples of fish, mainly eel, from the Rhine and Neckar rivers exhibited TEQ concentrations in a range of 8.1–39.2 pg/g fat. The maximum level was found in a barbel sample with a relatively low fat content (58). Samples of bream from the river Elbe (59) can in part be characterized as highly contaminated (4.1–23.8 pg TEQ/g fresh weight, 1% fat content). Levels were highest in fish from the harbor of Hamburg (up to 95 pg/g for 2,3,7,8-TCDD), presumably because of high levels in sediments (60).

Food from contaminated areas near PCDD/PCDF-emitting plants exhibited elevated levels as shown by samples of cow's milk from different industrialized areas (61) as well as by samples from geese and kale from the surroundings of a metal reclamation plant. Levels in adipose tissue from geese sampled in the contaminated area were 20 times higher than in the reference samples with background contamination (Table 14). The respective differences for kale were not so clear (Table 15). Even the samples with background contamination showed comparably high levels, possibly due to the long period of growth, which extends into the cold-weather months (55).

Other data (57) did not show elevated levels in samples of cow's milk collected near a municipal waste incinerator.

Prinz et al. (62,63) compared levels in potatoes, salad, carrots, celery, chard, leek, cucumbers, beans, plums, strawberries, apples, beetroot, kohlrabi, savoy cabbage, endive, and kale in the vicinity of a cable waste incinerator with samples from a rural area taking into account the levels in soil. The calculated transfer rates for leafy vegetables were higher than those for root vegetables, fruit vegetables, and fruits. The transfer from soil into plants seems to be relatively low.

A comparison of concentrations in soil and corn and in soil and grass, respectively, revealed a low correlation. Contamination through ambient air pollution and air-plant transfer predominates (62,63–65). In all food samples of vegetable origin, the congener pattern distribution resembles that of general combustion with a dominance of non-2,3,7,8-substituted congeners.

Körner et al. (66) investigated the PCDD/PCDF formation in smoked, fried, and broiled meat and fish. For cold smoked meat as the main source among this group, 2.9 pg TEQ/person/day was calculated as an additional intake. The congener patterns were characteristic for PCDD/PCDF formation under conditions of

incomplete combustion with a dominance of non-2,3,7,8-substituted congeners.

After findings of PCDDs/PCDFs (especially 2,3,7,8-TCDD/TCDF) in paper products (67,68) the carry-over of these substances into food was investigated for cow's milk packaged in cardboard containers (61) and brewed coffee prepared with coffee-filter paper (69). For milk samples packaged in cardboard containers, a transfer of about 10% for TEQs from paper into milk was determined during a 6-day storage (57,61,70). The levels increased during this period (70). After the PCDD/PCDF levels in cardboard containers used in Germany had been distinctly reduced (≤ 1 ppt TEQ in paper) by the manufacturers, the mean daily intake of TEQs by cardboard containers with 0.01 pg TEQ/kg body weight per day will now contribute less than 1% as compared to the total intake via food. Levels in coffee-filter paper also have been reduced. Lower levels for this item are 0.2 pg/g for 2,3,7,8-TCDD and 1 pg/g for 2,3,7,8-TCDF (Beck et al., unpublished results), also resulting in a share of less than 1% compared to the total intake of food.

Air

In comparison to the intake via food, the intake via outdoor air is considered to be relatively low (26). Assuming a TEQ level of 0.2 pg/m³ (51,54), a complete absorption and a respiration volume of 20 m³/day, the daily intake is calculated to be 4 pg TEQ/person/day and 0.06 pg TEQ/kg body weight per day, respectively. This is only 2% of the daily intake calculated for food. So only in special cases with relatively high levels of air contamination [e.g., caused by indoor use of PCP for wood preservation (51,54)] the intake for TEQs via long-term inhalation would become important.

Coplanar and Mono-Ortho-Substituted PCBs

Because several *in vitro* investigations have shown similar toxicological properties of coplanar (non-ortho) and mono-ortho-substituted PCB congeners to PCDD/PCDF, these compounds have also been measured in human samples.

Beck et al. (71) reported results of the analysis of milk and adipose tissue samples of human origin for 3,3', 4, 4'-tetrachlorobiphenyl (PCB-77): Mean levels were 22 and 51 pg/g fat, respectively. The other coplanar congeners [3,3', 4,4', 5-pentachlorobiphenyl (PCB-126) and 3,3', 4,4', 5,5'-hexachlorobiphenyl (PCB-169)] were not quantified exactly, but it was estimated that their levels according to literature data (72,73) were higher by factors of approximately 4 and 2, respectively.

PCB-77 contributes only 0.001% to total PCBs in human samples, whereas its level in some technical products exceeds 0.1% (74). This means that this congener is less resistant to biodegradation than many other higher chlorinated congeners. In the technical products, the other non-ortho-substituted congeners (PCB-126 and PCB-169) were determined in concentrations distinctly lower than that of PCB-77 (74).

In human milk, among the mono-ortho-substituted PCB congeners, 2,3',4,4',5-pentachlorobiphenyl (PCB-118) was mainly present in concentrations of about 60 ng/g milk fat as the main congener. The other congeners of this group [2,3',4',5-tetrachlorobiphenyl (PCB-70), 2,3,3',4,4'-pentachlorobiphenyl

(PCB-105), 2,3,3',4,4',5-hexachlorobiphenyl (PCB-156), 2,3,3',4,4',5'-hexachlorobiphenyl (PCB-159)] were estimated to be in lower concentrations in a range between 1 and 30 ng/g milk fat (75–77 and H. Beck, unpublished results).

Conclusions

For nonoccupationally exposed persons, the daily intake via food consumption is relatively high. Other sources and pathways are of minor importance. Food of animal origin contributes most, although human exposure begins with atmospheric emissions depositing these compounds on plant surfaces or on soil. Therefore, efforts should be made to minimize or avoid PCDD/PCDF emissions into the environment. Especially, air concentrations of these substances must be reduced to decrease the food chain accumulation (56), leading at present to levels in human milk not tolerable under the aspect of prospective health care.

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